MYOCARDIAL INFARCTION

BY

T. E. LOWE AND W. B. WARTMAN

From the Department of Pathology, University of Melbourne, and the Institute of Pathology, Western Reserve University and University, Hospitals of Cleveland, Cleveland, Ohio

Owing to postal delays caused by the authors' movements, this summary of their recent paper (British Heart J., **6,** 115–128, 1944) *was not received in time to print with the paper. The opportunity is taken of adding one sentence and correcting two misprints.*—EDITOR.

Any discussion of myocardial infarction should correlate the pathological changes with the anatomical structure of the heart. In this paper a review of the anatomy of the cardiac muscles and of the coronary arterial tree and the diseases thereof that may lead to muscle infarction is given. The consequences of interference with the blood flow in this tree are discussed.

The ventricular and auricular (atrial) walls are composed of several muscles, which are in reality as distinct as the three glutei muscles making up the gluteal mass. The separation of these muscles was first shown by anatomical dissection, and has been confirmed by the demonstration of independent blood supplies, and their individual involvement in various pathological states.

Anastomotic channels of arterial size between major coronary arteries exist of all ages. Pressure difference between the ends of any vessel is the factor which determines the direction of blood flow in that vessel. In a normal coronary circulation arterial anastomoses will have approximately the same pressure at either end, so that the blood supply will be small and may easily change in direction from time to time. In hearts the seat of obstructive arterial changes, marked difference of pressure between the ends of the anastomotic channels may easily occur, and then these vessels enlarge and become visible to the naked eye. The distribution of fields of supply will also become upset and it will then be impossible to predict the field of supply of any one of the major coronary arteries.

No one arterial branch supplies the whole of any one ventricular muscle. It is important that the parent artery, which gives branches to the pericardial portion of a superficial muscle, gives branches also to the endocardial portion of that muscle, but not to the deep branches in between.

No essential difference exists between diseases of the arteries of the heart and those of other organs of the body. Regardless of which portion of the coronary tree is affected, the gravity of the lesion is largely determined by the degree of vascular obstruction. In most cases thrombosis is secondary to underlying vascular disease, and primary thrombosis of the coronary arteries has not been encountered. A discussion of the various diseases of the coronary arteries follows. The importance of arteriosclerotic intimal hæmorrhage in precipitating thrombosis and vascular occlusion is stressed. Similar hæmorrhages in the media lead to dissecting aneurysm.

Myocardial ischæmia is discussed under the following headings: (1) infarction of the ventricular wall; (2) interference with ventricular function; (3) rupture of the ventricle; (4) ventricular aneurysms; (5) infarction of the atrial wall; and (6) electrocardiographic changes. The effects of sudden and gradual occlusion in normal and diseased coronary

arteries are considered. The resulting infarcts may involve parts of several muscles (whole thickness of ventricular wall), or may be confined to a portion of one muscle, or may form scattered small foci.

Interference with ventricular function may result either from an inability of the ischæmic heart adequately to expel blood or from the development of abnormal rhythms.

The physical basis of aneurysm formation is the same in both ventricle and aorta, for in both places muscle tissue is active in withstanding pressure, and when enough of it is destroyed and replaced by collagen, gradual stretching and sac formation occurs. Two types of ventricular aneurysms are described. Basal aneurysms show two laminar scars indicating that destruction of two deep muscles is essential to their formation. Apical aneurysms, in contrast, show a single laminar scar due to the involvement of the superficial musculature.

The principles outlined in discussing ventricular infarcts are strictly applicable to those occurring in the walls of the auricles. Auricular infarcts occur in about 17 per cent of cases of ventricular infarction and usually involve the right auricular appendage. Disturbance in auricular electrical activity in such cases may suggest damage of the auricular muscles, possibly due to infarction.

The paper concludes with a brief discussion of the limitations of electrocardiographic changes.

ERRATA

Page 119.

Line 6. *Insert* " In most cases thrombosis is secondary to underlying vascular disease, and hence may complicate any of the lesions described below. We have not encountered primary thrombosis of the coronary arteries."

Line 16. *Insert* " by " between " muscle " and " fibrous tissue."

Line 19. *Insert* " 20 " for " 10 " before per cent.

Corrected References

Flett, R. L. (1927). *J. Anat.*, **62**, 439.
Wartman, W. B. (1933). *Amer. J. med. Sci.*, **197**, 7.
—— (1940). *American Association for the Advancement of Science*, Publication 13.